MECHANISM OF MUSCLE CONTRACTION

The way muscles tighten is described by a theory known as the sliding filament theory. This idea was put forward by two groups of researchers (Andrew Huxley and Ralph Niedergerke, Hugh Huxley and Jean Hanson) in 1954. As per this theory, muscles contract when a thin thread called actin slides over a thick thread called myosin.

- The tightening of muscles begins when the central nervous system (CNS) sends a signal through a motor neuron. This nerve message from the CNS travels along the motor neuron. Once it reaches the end of the neuron, called the axon terminal or neuromuscular junction, small sacs with neurotransmitters join with the axon's outer layer. After they fuse with the axon membrane, they release a neurotransmitter called acetylcholine. This acetylcholine moves across a tiny gap called the synaptic cleft and causes a change in electrical charge on the muscle's outer layer, called the sarcolemma. The normal electrical charge across the membrane of a relaxed muscle fiber, known as resting potential, is approximately –90 mV.
- The signal or action potential that occurs then moves from the outer layer of the muscle (sarcolemma) to the T-tubules. This signal then prompts the sarcoplasmic reticulum to let go of calcium ions into the sarcoplasm.
- When there's more calcium in the sarcoplasm, it kickstarts the sliding of filaments. On the flip side, a decrease in calcium shuts down the sliding process.
- When a muscle fiber is not contracting, it means it's relaxed, and the amount of Ca^{2+} in its sarcoplasm is low. This happens because the membrane of the sarcoplasmic reticulum (SR) has active transport pumps for Ca^{2+} that move it from the sarcoplasm into the SR, where it's stored. When a muscle action potential travels along the sarcolemma into the transverse tubule system, channels in the SR membrane that release Ca^{2+} open up. This causes a flood of Ca^{2+} into the sarcoplasm around the thick and thin filaments. The released Ca^{2+} combines with troponin, making it change shape. This change in shape shifts the troponin tropomyosin complex away from the myosin-binding sites on actin.

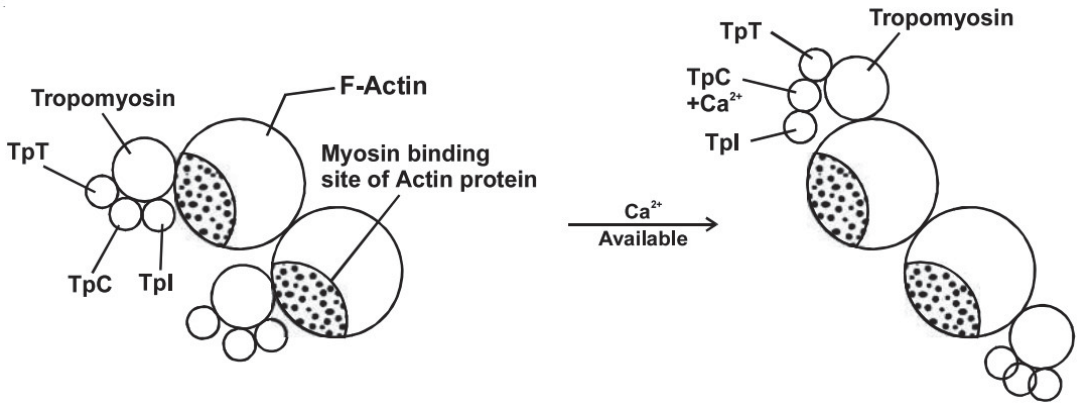
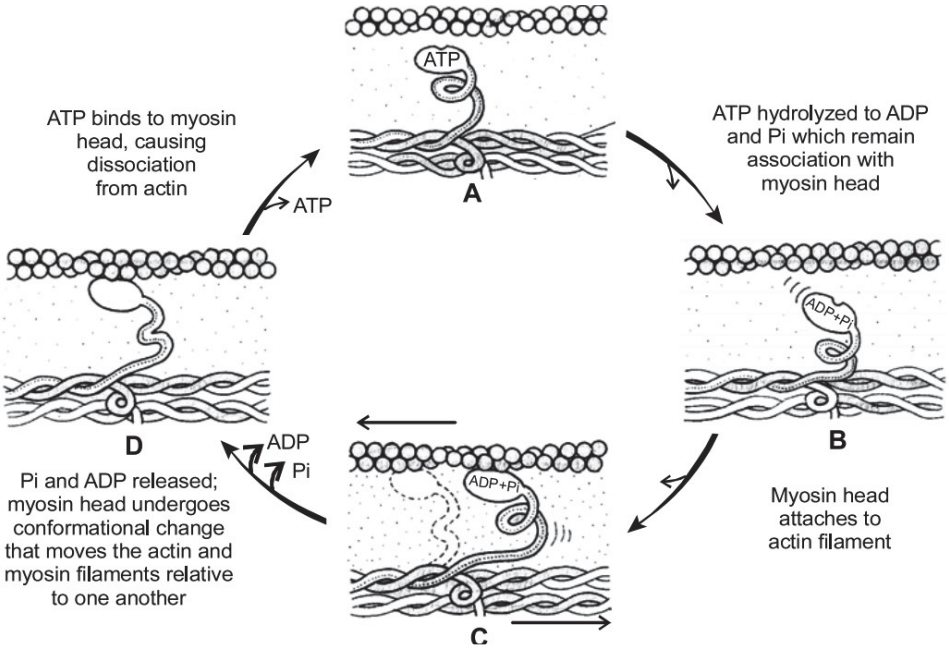
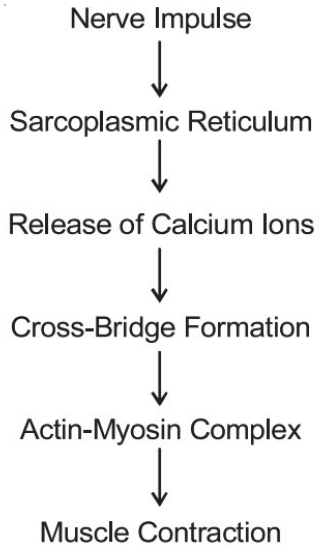


Fig.: Role of Ca^{2+} in muscle contraction by exposing myosin binding site of actin protein

- The round head of myosin works like an ATPase, breaking down ATP molecules. The energy released from this breakdown is used by myosin to connect with the open active site on the actin filament, creating a cross-bridge.



- This pulls the attached actin filament toward the center of the 'A-band.' The Z-line connected to these actins is also drawn inward, leading to a shortening of the sarcomere, or contraction. The thin myofilaments move past the thick myofilament, making the H-zone narrower and increasing the overlap zone. This shortens the I-band but keeps the A-band length unchanged. Afterward, myosin releases ADP+Pi and returns to its relaxed state. Once again, ATP attaches to myosin, breaking the connection or cross-bridge between myosin and actin. ATP is then broken down, and the cycle of forming and breaking cross-bridges is repeated, causing sliding. This process continues until calcium ions are pumped back into the sarcoplasmic reticulum.
  This causes the return of 'Z' lines back to their original position i.e., relaxation occurs.



Flow chart of Muscle Contraction